

Automated Peritoneal Dialysis is Suitable for Polycystic Kidney Disease Patients with End-Stage Renal Disease

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Key Words

Polycystic kidney disease · End-stage renal disease · Renal replacement therapy · Automated peritoneal dialysis

Abstract

A female patient with polycystic kidney disease (PKD) was treated with automated peritoneal dialysis when she reached end-stage renal disease. The patient has been doing very well on automated peritoneal dialysis (APD) for almost 6 years without peritonitis or abdominal hernias. Intra-abdominal pressures are lower in the supine position than in an erect or sitting position. Larger volumes of dialysate are better tolerated while the patient is supine, as during nocturnal APD. Therefore, APD is an option of the renal replacement therapy for patients with PKD.

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Introduction

Autosomal polycystic kidney disease (PKD) is the most common monogenic renal disease [1]. There are about 400–600 million PKD patients all over the world. The United States renal data system (USRDS) reports that 20,000 patients with PKD progress into end-stage renal disease (ESRD) each year in the United States. By the end of 2011, a total of 395 patients with PKD were undergoing dialysis in Shanghai; this accounted for 3.15% of all dialy-

sis patients, only less than glomerulonephritis, diabetic nephropathy and hypertensive renal sclerosis [2].

What's the better option in renal replacement therapy for PKD patients with ESRD, peritoneal dialysis (PD) or hemodialysis (HD)? The answer is quite controversial. We introduced a patient with PKD, treated by automated peritoneal dialysis (APD).

Case Presentation

A female, born in 1965, was admitted to our Nephrology Department in December 2008.

History of Present Illness

PKD was found 20 years ago via a physical examination (with no specific treatment). Her annual physical examination showed a normal serum creatinine level. In January 2007, her serum creatinine level was elevated to 247 $\mu\text{mol/l}$. The patient was treated with anti-hypertensive medications. She received a renal cyst puncture and ethanol injection at the other hospital on August 29, 2008. She suffered from a cyst infection with high fever; she was then given positive anti-infective therapy. Her body temperature returned to normal while her renal function deteriorated further. The serum creatinine level was 648 $\mu\text{mol/l}$, albumin was 33 g/l, and hemoglobin was 87 g/l before the admission. A renal replacement therapy was considered.

Past Medical History

She had hypertension for 10 years. She suffered from a cerebral hemorrhage in 2001, which caused weakness in her right extremities.

Personal History

Our patient is married. She has a healthy daughter and denies any tobacco and alcohol addiction. Her mother and an uncle suffered from PKD.

Physical Examination

Her body temperature was 36°C, pulse 80/min, respiratory rate 20/min, blood pressure 110/60 mm Hg, and body surface 1.67 m². The patient appeared to be oriented and cooperative. She looked pale. She had a normal skin appearance, texture, and temperature. No palpable nodes were in the cervical, supra-clavicular, axillary or inguinal areas. Her oral pharynx was normal without erythema or exudate. The neck was easily movable without any resistance, no abnormal adenopathy in the cervical or supraclavicular areas was observed. The trachea was midline and the thyroid gland was normal without any visible masses. Her lungs were clear to auscultation and percussion bilaterally. The heart rate was 80 bpm with a regular rhythm and no murmur. The abdomen was distended; bowel sounds were normal. Her enlarged kidneys were palpable and no edema was found in both lower extremities; her muscle strength of the right limbs was grade IV.

Laboratory Findings

Full blood count was as follows: white blood cells $5.4 \times 10^9/\text{l}$, red blood cells $3.20 \times 10^{12}/\text{l}$, hemoglobin 95 \downarrow g/l, platelets $216 \times 10^9/\text{l}$. Fast blood glucose 4.90 mmol/l, alanine aminotransferase 9 IU/l, glutamic oxalocetic transaminase 12 IU/l, prealbumin 208 mg/l, alkaline phosphatase 47 IU/l, r-GT 15 IU/l, total bilirubin 13.1 $\mu\text{mol/l}$, direct bilirubin 1.6 $\mu\text{mol/l}$, total protein 73 g/l, albumin 36 g/l, blood urea nitrogen 24.0 \uparrow mmol/l, serum creat-

inine 731.0 \uparrow $\mu\text{mol/l}$, uric acid 390 $\mu\text{mol/l}$, triglyceride 1.20 mmol/l , total cholesterol 3.94 mmol/l , sodium 138.5 mmol/l , potassium 4.45 mmol/l , calcium 2.32 mmol/l , phosphorus 1.68 mmol/l , ferritin 57.8 ng/ml , transferrin 189 mg/dl , serum ferritin 7.6 $\mu\text{mol/l}$, iron saturation 17.0%, total iron binding capacity 44.8 $\mu\text{mol/l}$, parathyroid hormone 131.2 pg/ml . A B-ultrasound scan showed intrahepatic cystic lesions and liver cysts should be considered; the kidneys were significantly enlarged (occupied in the upper abdomen, down to the pelvis), consistent with polycystic kidney disease. Electrocardiogram was normal. Echocardiography: ejection fraction was 62%. A head enhanced CT scan performed in another hospital showed a possible cerebral aneurysm.

Treatment

Losartan (50 mg 1 tablet, once a day, p.o.), levamlodipine (2.5 mg 1 tablet, once a day, p.o.), clonidine (75 μg 1 tablet, three times a day, p.o.), calcitriol (0.25 μg 1 tablet, once a day, p.o.), calcitrate D (0.6 g 1 tablet, once a day, p.o.), bicarbonate (500 mg 2 tablets, three times a day, p.o.), ferrous succinate (100 mg 2 tablets, three times a day, p.o.). A laparoscopic peritoneal dialysis catheter insertion was performed on December 9, 2008, and automated peritoneal dialysis was started after 2 weeks. The treatment protocol consisted of 1.5% peritoneal dialysate (8 liters, 2 liters \times 4 cycles in a total of 10 h; nocturnal). The patient has been doing very well on APD for almost 6 years (table 1). She has never suffered from peritonitis or abdominal hernias.

Discussion

It is commonly believed that PKD patients on PD are overexposed to technique failure and peritonitis compared to other patients. Extrarenal manifestations of PKD, such as diverticulosis, development of hernias or vascular aneurysms, may theoretically promote the occurrence of complications typically related to PD. Nevertheless, enlarged kidneys occupying most of the abdominal cavity space might reduce the available peritoneal surface area, restricting the dialysis volume. Thus, most patients with PKD progressing to ESRD are usually referred to hemodialysis. The report of Shanghai Renal Registry [2] shows that patients with ESRD caused by PKD are less frequently treated with PD than patients with noncystic renal diseases (7.5 vs. 24.1%). In contrast, USRDS reports that more than 20% of PKD patients choose PD as their initial form of renal replacement therapy, while only 15% are shown in non-PKD renal failure patients [3].

Is peritoneal dialysis appropriate for PKD patients with ESRD? Hadimeri et al. [4] compared survival and complications between 26 patients with autosomal dominant polycystic kidney disease (ADPKD) under continuous ambulatory peritoneal dialysis (CAPD) and 26 controls without ADPKD or diabetic nephropathy. They found that it is similar in survival rate in ADPKD patients and controls. Hernia was present in 4 ADPKD patients and 2 controls, and 1 patient was required to transfer to HD temporarily in each group. The incidence of peritonitis was 1 in 20 months in ADPKD patients versus 1 in 27 months in controls (not significantly different). Peritonitis caused by colonic bacteria was in similar numbers. Residual renal function was similar in ADPKD patients and controls. No difference was detected in any of the variables measured by peritoneal dialysis capacity. A retrospective cohort study [5] based on the data of the French Language Peritoneal Dialysis Registry also suggested that PD is a suitable method for at least a subgroup of PKD patients reaching end-stage renal disease in a country where renal transplantation is available.

Xie et al. [6] retrospectively analyzed 29 patients with PKD who needed dialysis therapy for over 3 months in our department. They were treated with CAPD (n = 10) and HD (n = 19), and 10 CAPD patients with non-PKD served as controls. No significant difference existed in the survival rates of 1-, 3- and 5-years among 3 groups. Kt/V per week, creatinine clearance, residual renal function, peritoneal transport characteristics, 24h urine and ultrafiltration were similar between PKD and non-PKD CAPD patients. No patient changed to HD due to inadequate PD dialysis. We concluded that the prognosis and complication incidence in PKD and non-PKD patients treated with CAPD are similar, and the prognosis of PKD patients treated with CAPD or HD is also similar.

Abdominal hernias and peritoneal leaks are very frequent in the PD population. Advanced age, PKD and a high body mass index are independent risk factors for their development [7]. However, there are some advantages of APD in patients with PKD. The patient with PKD introduced above has been doing very well on APD for almost 6 years. She has never suffered from peritonitis or abdominal hernias. Because intra-abdominal pressures are lower in the supine position than in the erect and sitting position, larger volumes of dialysate are better tolerated while the patient is supine (as during nocturnal APD) [8]. The above patient has excellent residual renal function (renal creatinine clearance was between 52.51 and 62.38 liters/week) and well controlled in blood pressure. Increased and individualized dwell volumes and prescriptions for suitable total volumes per session in APD permit longer dwell times and allow for better results in patients with a peritoneal permeability lower than the mean. In order to avoid peritoneal leaks and hernia, APD treatment with low daytime exchange volumes must be considered in those patients with PKD [7].

In conclusion, the technical survival, quality of dialysis, duration of therapy and rates of complications in PD are comparable in patients with cystic or noncystic kidney disease, and therefore all patients with PKD who do not have abdominal complaints or a history of recurrent hernias should be informed that PD is an adequate form of renal replacement therapy. Moreover, APD is a suitable method of renal replacement therapy for PKD patients with ESRD due to its flexibility and effectiveness.

Disclosure Statement

On behalf of Dr. Chen Nan and the other authors, we declare that no financial or other conflict of interest exists in relation to the content of the paper.

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Table 1. Dialysis adequacy parameters after starting APD

Months after APD	Kt/V, /week	Ccr, l/week	24 h urine, ml
1	2.98	75.20	1,400
6	3.12	84.02	2,000
12	3.3	88.16	1,400
54	4.25	90.06	1,500
70	2.38	48.89	1,100

Ccr = Creatinine clearance.